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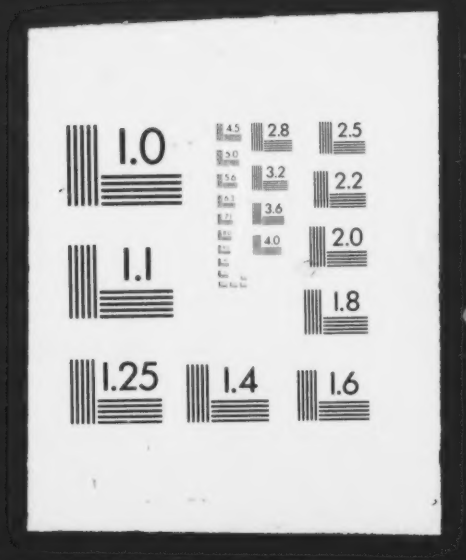

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Health and Environmental Effects Profile for Methyl Acetate

(U.S.) Environmental Protection Agency
Cincinnati, OH

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16. ABSTRACT

The Health and Environmental Effects Profile for Methyl Acetate was prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste and Emergency Response to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA) and to provide health-related limits for emergency actions under Section 101 of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). Both published literature and information obtained from Agency program office files were evaluated as they pertained to potential human health, aquatic life and environmental effects of hazardous waste constituents. Quantitative estimates have been presented provided sufficient data are available. Methyl acetate has been determined to be a systemic toxicant. A Reference Dose (RfD), defined as an estimate (with uncertainty spanning perhaps an order of magnitude) of the daily exposure to the human population (including sensitive subgroups) that is likely to be without appreciable risk of deleterious effect during a lifetime, for methyl acetate is 1 mg/kg/day for oral exposure. The Reportable Quantity (RQ) value of 1, 10, 100, 1000 or 5000 pounds is used to determine the quantity of a hazardous substance for which notification is required in the event of a release as specified by CERCLA based on chronic toxicity. Existing data are insufficient to determine an RQ value.

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HEALTH AND ENVIRONMENTAL EFFECTS PROFILE FOR METHYL ACETATE

ENVIRONMENTAL CRITERIA AND ASSESSMENT OFFICE
OFFICE OF HEALTH AND ENVIRONMENTAL ASSESSMENT
OFFICE OF RESEARCH AND DEVELOPMENT
U.S. ENVIRONMENTAL PROTECTION AGENCY
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PREFACE

Health and Environmental Effects Profiles (HEEPs) are prepared for the Office of Solid Waste and Emergency Response by the Office of Health and Environmental Assessment. The HEEPs are intended to support listings of hazardous constituents of a wide range of waste streams under Section 3001 of the Resource Conservation and Recovery Act (RCRA), as well as to provide health-related limits for emergency actions under Section 101 of the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA). Both published literature and information obtained from Agency program office files are evaluated as they pertain to potential human health, aquatic life and environmental effects of hazardous waste constituents. The literature searched and the dates of the searches are included in the section titled "Appendix: Literature Searched." The literature search material is current through November, 1985.

Quantitative estimates are presented provided sufficient data are available. For systemic toxicants, these include Reference doses (RfDs) for chronic exposures. An RfD is defined as the amount of a chemical to which humans can be exposed on a daily basis over an extended period of time (usually a lifetime) without suffering a deleterious effect. In the case of suspected carcinogens, RfDs are not estimated in this document series. Instead, a carcinogenic potency factor of q_1^* is provided. These potency estimates are derived for both oral and inhalation exposures where possible. In addition, unit risk estimates for air and drinking water are presented based on inhalation and oral data, respectively.

Reportable quantities (RQs) based on both chronic toxicity and carcinogenicity are derived. The RQ is used to determine the quantity of a hazardous substance for which notification is required in the event of a release as specified under CERCLA. These two RQs (chronic toxicity and carcinogenicity) represent two of six scores developed (the remaining four reflect ignitability, reactivity, aquatic toxicity and acute mammalian toxicity).

The first draft of this document was prepared by Syracuse Research Corporation under EPA Contract No. 68-03-3228. The document was subsequently revised after reviews by staff within the Office of Health and Environmental Assessment: Carcinogen Assessment Group, Reproductive Effects Assessment Group, Exposure Assessment Group, and the Environmental Criteria and Assessment Office in Cincinnati.

The HEEPs will become part of the EPA RCRA and CERCLA dockets.

ACKNOWLEDGEMENTS

The initial draft of this report was prepared by Syracuse Research Corporation under Contract No. 68-03-3228 for U.S. EPA's Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH. Dr. Christopher DeRosa was the Technical Project Monitor and Helen Ball was the Project Officer. The final documents in this series were prepared for the Office of Solid Waste, Washington, DC.

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EXECUTIVE SUMMARY

Methyl acetate is a colorless, volatile liquid that is miscible in common organic solvents and is soluble in water (Windholz, 1983; Hawley, 1981). Current manufacturers include Monsanto Chemical and Borden, Inc. (SRI, 1986). Methyl acetate can be produced directly by reacting methanol and acetic acid (Hawley, 1981), but it is also formed as a by-product during acetic acid manufacture by n-butane oxidation (Soder, 1976). According to the public portion of the U.S. EPA TSCA Production File for 1977, total production exceeded 21 million pounds (U.S. EPA, 1977). The use pattern for methyl acetate has been reported as follows (Wagner, 1978): paint remover, 85%; lacquer solvent, 12%; chemical raw material, 3%.

When released to the aquatic environment, microbial degradation and volatilization are likely to be important removal mechanisms for methyl acetate. Based on limited experimental data and by analogy to ethyl acetate, methyl acetate appears to be susceptible to microbial degradation in the environment; U.S. EPA (1986a) suggested that methyl acetate should biodegrade moderately fast. The volatilization half-life from a river 1 m deep flowing at a speed of 1 m/sec with a wind velocity of 3 m/sec has been estimated to be 10.8 hours. Hydrolysis will not be important in neutral or acidic water, but may become significant in alkaline water; the respective hydrolysis half-lives at 25°C and pHs 7, 8 and 9 are 1.2 years, 45 and 4.5 days. Aquatic oxidation (by HO radical), adsorption to sediments and bioconcentration are not expected to be important. If released to the atmosphere, methyl acetate will react with photochemically produced HO radical with a half-life of ~59 days at 19°C. If released to soil, methyl acetate is likely to be susceptible to significant leaching and biodegradation. Volatilization from dry surfaces is expected to be rapid.

Methyl acetate occurs naturally in volatile emissions from animal wastes and forest fires (Graedel, 1978). It has been qualitatively detected in drinking water from Seattle, WA (U.S. EPA, 1975) and in wastewater effluents from chemical manufacturing and latex facilities in Louisville, KY (U.S. EPA, 1976). Direct evaporation of methyl acetate solvent from paint removers and lacquers is probably the largest volume source of emission of the compound to the environment. Methyl acetate has been detected in the air of the Talladega National Forest, AL (Holzer et al., 1977) and in cigarette smoke (Higgins et al., 1983).

Pertinent data regarding the pharmacokinetics and metabolism of methyl acetate are limited. After oral administration to rabbits, methyl acetate was hydrolyzed to methanol and acetic acid (Tambo, 1973). Tada et al. (1974) exposed humans to 200 ppm (607 mg/m^3) methyl acetate, by inhalation, twice daily for 2 hours each for 3-4 days. Urinary excretion of methanol rose during the first daily exposure, peaked during the second exposure and returned to preexposure levels the following morning. Maximum urinary concentrations of methanol were generally >10 mg/ℓ.

There were no data available on the carcinogenic, reproductive, chronic or subchronic effects of methyl acetate administration. Acutely, the compound is a respiratory and mucous membrane irritant (Reus, 1933). In cats, inhalation exposure for 5 days resulted in increased hemoglobin and erythrocyte levels, and a transient leukocytosis (Reus, 1933). Toxic effects after acute inhalation exposure also included a gradual narcosis and metabolic acidosis (Fassett, 1963). Methyl acetate produced erythema when applied to rabbit skin, and hyperemia and edema when applied to the eye (von Oettinger, 1960). NIOSH (1977) stated that the lowest toxic concentration in man was 15,000 mg/m^3.

Studies regarding the mutagenicity of methyl acetate are limited to the yeast Saccharomyces. Zimmermann et al. (1985) found that methyl acetate induced mitotic aneuploidy but not mutation or recombination in Saccharomyces cerevisiae. Methyl acetate may produce chromosomal malsegregation by interfering with tubulin assembly (Groschel-Stewart et al., 1985). Abbondandolo et al. (1980) found that methyl acetate was more lethal to V79 Chinese hamster cells than to Schizosaccharomyces pombe cells in culture. In a hypotonic solution of rat erythrocytes, the ester exerted an anti-hemolytic effect (Holmberg et al., 1974).

The only information available concerning the effects of methyl acetate on aquatic organisms was provided by Munch (1972), who reported threshold narcotic concentrations of 70-157 mmol/ℓ (5186-11,631 mg/ℓ) for tadpoles, Rana pipiens, and by Calmbacher (1978), who reported that the 96-hour LC_{50} for bluegills, Lepomis macrochirus, was >100 mg/ℓ.

An RfD of 1 mg/kg/day or 81 mg/day for a 70 kg man was derived by analogy to methanol because toxic effects of methyl acetate are attributed to its hydrolysis product, methanol. The RfD for methanol was based on an oral subchronic rat NOEL of 500 mg/kg/day in a study by Toxicity Research Laboratory (1986), using an uncertainty factor of 1000. The NOEL for methanol was corrected for molecular weight differences to give a NOEL of 1156 mg/kg/day for methyl acetate. Data were insufficient for deriving an RQ. Data regarding the carcinogenicity of methyl acetate are not available; therefore, methyl acetate is classified as a Group D chemical according to EPA's Carcinogen Risk Assessment Guidelines (U.S. EPA, 1986d) (i.e., not classifiable as to human carcinogenicity).

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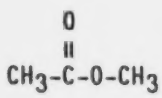
LIST OF ABBREVIATIONS

ADI	Acceptable daily intake
BCF	Bioconcentration factor
CAS	Chemical Abstract Service
CNS	Central nervous system
K_{ow}	Octanol/water partition coefficient
LC_{50}	Concentration lethal to 50% of recipients (and all other subscripted dose levels)
LD_{50}	Dose lethal to 50% of recipients
LOAEL	Lowest-observed-adverse-effect level
NOEL	No-observed-effect level
ppm	Parts per million
RfD	Reference dose
RQ	Reportable quantity
SGPT	Serum glutamic oxaloacetic transaminase

1. INTRODUCTION

1.1. STRUCTURE AND CAS NUMBER

Methyl acetate is the common chemical name, but this compound is also known as methyl ethanoate and acetic acid, methyl ester. The structure, molecular weight, empirical formula and CAS Registry number are as follows:



Molecular weight: 74.08

Empirical formula: $C_3H_6O_2$

CAS Registry number: 79-20-9

1.2. PHYSICAL AND CHEMICAL PROPERTIES

Methyl acetate is a colorless, volatile liquid with a pleasant odor (Windholz, 1983; Hawley, 1981). It is miscible with alcohol, ether and other common hydrocarbon solvents and is soluble in water (Windholz, 1983; Hawley, 1981). Selected physical properties are listed below:

Melting point:	-99°C	Verschueren, 1983
Boiling point:	56.9°C	Windholz, 1983
Specific gravity:	0.9279 (25/4°C)	Windholz, 1983
Refractive index:	1.3614 (20°C)	Windholz, 1983
Vapor pressure:		
at 20°C	173 mm Hg	Union Carbide, 1973
at 25°C	217 mm Hg	Boublik et al., 1984
Vapor density:	2.56	Verschueren, 1983
Water solubility:		
at 20°C	24.5 wt %	Union Carbide, 1973
at 22°C	33 g/100 g water	Perry and Green, 1984
at 25°C	330,000 ppm	U.S. EPA, 1986a
Log K_{ow}:	0.18	Hansch and Leo, 1985

Flash point:	-10°C (closed cup)	Windholz, 1983
Autoignition temperature:	501°C	Hawley, 1981
Conversion factors (air): at 20°C:	1 mg/m³=0.33 ppm 1 ppm = 3.03 mg/m³	Verschueren, 1983

Methyl acetate is flammable and a dangerous fire and explosion risk (Hawley, 1981); the explosive limits in air range from 3-16%.

1.3. PRODUCTION DATA

Methyl acetate is currently manufactured by Borden Inc., Compton, CA, and Illiopolis, IL, and by Monsanto, Springfield, MA (SRI, 1986). Monsanto and Eastman Kodak were reported as producers of this compound in 1984 by (USITC, 1985). The most recent production figure released for methyl acetate is 4.466 million pounds in 1977 (USITC, 1978). The approximate U.S. production capacity for methyl acetate has been reported to be 11 million pounds (Wagner, 1978), but this probably refers to purified methyl acetate intended for end-use and does not include materials produced as by-products. Table 1-1 lists production information available from the public portion of U.S. EPA TSCA Production File for 1977 (U.S. EPA, 1977). According to Table 1-1, a minimum of 21 million pounds of methyl acetate were produced in 1977, which is substantially higher than the 4.466 million pound figure reported by USITC (1978) for the same year; however, the USITC (1978) production figure probably does not include the by-product material.

Methyl acetate is directly manufactured by heating methanol and acetic acid in the presence of sulfuric acid and distilling (Hawley, 1981). It is formed as a by-product during the commercial production of acetic acid by the liquid-phase oxidation of n-butane (Soder, 1976); Celanese Corp. in Pampa, TX, and Union Carbide in Brownsville, TX, produce acetic acid by this method (SRI, 1986; Soder, 1976). It is also formed as a by-product from wood carbonization and can be recovered on a commercial basis (McNeil, 1983).

TABLE 1-1

U.S. Methyl Acetate Production Data for 1977[a]

Producer/Location	Manufacturer/ Importer	Production Volume (pounds)
Publicker Industries, Philadelphia, PA	manufacturer	confidential
Eastman Kodak, Rochester, NY	manufacturer	none
Fisher Scientific Co., Fair Lawn, NJ	manufacturer	confidential
DuPont, Wilmington, DE	manufacturer[b]	confidential
Union Carbide,		
Brownsville, TX	manufacturer	confidential
South Charleston, WV	manufacturer	confidential
Monsanto, Indian Orchard, MA	manufacturer	10-50 million
Tennessee Eastman, Kingsport, TN	manufacturer	1-10 million
Air Products & Chem., Calvert City, KY	manufacturer	confidential
Celanese Chem., Pampa, TX	manufacturer[b]	10-50 million
Henkel, Inc., Teaneck, NJ	importer	<1000
EM Laboratories, Elmsford, NY	importer	confidential
BASF Wyandette, Parsippany, NJ	importer	none
American Hoechst, Bridgewater, NJ	importer	confidential
Diamond Shamrock, Cleveland, OH	importer	10-100 thousand
Degussa Corp., Teterboro, NJ	importer	<1000
ICI Americas, Wilmington, DE	importer	none

[a]Source: U.S. EPA, 1977

[b]Used on plant site only

1.4. USE DATA

The use pattern for methyl acetate has been reported as follows (Wagner, 1978):

paint remover	85%
lacquer solvent	12%
chemical raw material	3%

Windholz (1983) reported that methyl acetate is used as a solvent for nitrocellulose, acetylcellulose and many oils and resins, and in the manufacture of synthetic leather. It has also been used as a synthetic flavoring (Hawley, 1981).

1.5. SUMMARY

Methyl acetate is a colorless, volatile liquid that is miscible in common organic solvents and is soluble in water (Windholz, 1983; Hawley, 1981). Current manufacturers include Monsanto Chemical and Borden, Inc. (SRI, 1986). Methyl acetate can be produced directly by reacting methanol and acetic acid (Hawley, 1981), but it is also formed as a by-product during acetic acid manufacture by n-butane oxidation (Soder, 1976). According to the public portion of the U.S. EPA TSCA Production File for 1977, total production exceeded 21 million pounds (U.S. EPA, 1977). The use pattern for methyl acetate has been reported as follows (Wagner, 1978): paint remover, 85%; lacquer solvent, 12%; chemical raw material, 3%.

2. ENVIRONMENTAL FATE AND TRANSPORT PROCESSES

2.1. WATER

2.1.1. Hydrolysis. The second-order rate constants for the acid- and base-catalyzed hydrolysis of methyl acetate at 25°C have been reported to be 1.133×10^{-4} M^{-1} sec^{-1} and 0.179 M^{-1} sec^{-1}, respectively (Mabey and Mill, 1978). A neutral hydrolysis rate constant at 25°C could not be located in the available literature; however, the neutral hydrolysis rate constant for ethyl acetate at 25°C has been reported to be 1.53×10^{-10} sec^{-1} (Mabey and Mill, 1978). Assuming a similar neutral hydrolysis rate constant for methyl acetate, the following hydrolysis half-lives at 25°C are calculated for methyl acetate at various pHs:

pH	Half-life
5	15 years
6	10.6 years
7	1.2 years
8	45 days
9	4.5 days

Therefore, the hydrolysis of methyl acetate in neutral or acidic waters in the environment will not be significant; however, hydrolysis may become significant if the water is sufficiently alkaline (pH $\geq$9). The hydrolysis of methyl acetate in water produces acetic acid and methanol (Bleier, 1984).

2.1.2. Oxidation. The rate constant for the reaction of methyl acetate with HO radical in water at room temperature has been reported to be ~1.2×10^{8} M^{-1} sec^{-1} (Dorfman and Adams, 1973). Assuming the HO radical concentration of sunlit natural water is ~10^{-17} M (Mill et al., 1980), a half-life of 18 years can be calculated, which indicates no environmental signficance.

2.1.3. **Photolysis.** Pertinent data regarding the photolysis of methyl acetate could not be located in the available literature as cited in the Appendix.

2.1.4. **Microbial Degradation.** Only limited microbial degradation data for methyl acetate were located in the available literature. Chou et al. (1979) found that methyl acetate is readily acclimated and degraded anaerobically by wastewater treatment involving methane fermentation. Marion and Malaney (1963) found by the Warburg technique that Alcaligenes faecalis bacteria isolated from activated sludge can bio-oxidize methyl acetate.

The similar compound ethyl acetate has been shown to be readily biodegradable by a number of studies involving activated sludge and sewage (Ettinger, 1956; Heukelekian and Gellman, 1951; Lamb and Jenkins, 1952; Price et al., 1974; Stover and Kincannon, 1983; Takemoto et al., 1981; Young et al., 1968). It was also found to be readily biodegradable in a natural river water (Lamb and Jenkins, 1952).

Based on the limited methyl acetate data available and by analogy to ethyl acetate, methyl acetate appears to be susceptible to microbial degradation in the environment. U.S. EPA (1986a) suggested that methyl acetate should biograde moderately fast.

2.1.5. **Volatilization.** Based on experimental equilibrium data (Hine and Mookerjee, 1975), the Henry's Law constant for methyl acetate at 25°C is ~8.9×10^{-5} atm-m^3/mol. This value of Henry's Law constant indicates that volatilization may be signficant from all environmental bodies of water, although it may not be rapid (Lyman et al., 1982). Based on the method outlined in Lyman et al. (1982), the volatilization half-life of methyl acetate from a shallow river 1 m deep flowing at a speed of 1 m/sec with a

wind velocity of 3 m/sec is calculated to be 10.8 hours. Therefore, volatilization may be one of the important loss processes from aqueous media.

2.1.6. Adsorption. Experimental adsorption data on aquatic particulate matter could not be located in the available literature; however, based on a relatively low log K_{ow} value of 0.18 (Hansch and Leo, 1985) and high water solubility of 24.5 wt % at 20°C (Union Carbide, 1973), methyl acetate is not expected to significantly partition from the water column to sediments.

2.1.7. Bioconcentration. BCFs can be estimated from the following recommended equations (Lyman et al., 1982):

$$\text{Log BCF} = 0.76 \log K_{ow} - 0.23 \qquad (2\text{-}1)$$

$$\text{Log BCF} = 2.791 - 0.564 \log \text{WS (water solubility in ppm)} \qquad (2\text{-}2)$$

Based on a log K_{ow} value of 0.18 (Hansch and Leo, 1985) and water solubility of 245,000 ppm at 20°C (Union Carbide, 1973), the BCF values calculated from Equations 2-1 and 2-2 are 0.8 and 0.6, respectively, indicating that bioconcentration in aquatic organisms is not expected to occur.

2.2. AIR

The rate constant for the vapor-phase reaction between methyl acetate and photochemically produced HO radical in the atmosphere has been experimentally measured to be 0.17×10^{-2} cm³/molecule-sec at 19°C (Campbell and Parkinson, 1978; Atkinson, 1985). Assuming an average ambient atmospheric concentration of 8×10^5 HO radical/cm³, the calculated half-life for this reaction is 59 days. U.S. EPA (1980) considered the concentration differences in HO radical during summer and winter and estimated that in January 9 days are required to consume 1% of the methyl acetate present at 40°N latitude while in July only 0.9 days are required to consume 1% by HO radical reaction.

2.3. SOIL

2.3.1. Microbial Degradation. Although soil microbial degradation studies could not be located in the available literature, the apparent susceptibility of methyl acetate to biodegradation (see Section 2.1.4.) suggests that biodegradation by soil microflora is likely to occur.

2.3.2. Leaching. Experimental soil leaching data could not be located in the available literature as cited in the Appendix. The relatively high water solubility (24.5 wt % at 20°C) (Union Carbide, 1973) and low log K_{ow} value (0.18) (Hansch and Leo, 1985) of methyl acetate suggest that significant leaching may occur in many soil types.

2.3.3. Volatilization. Methyl acetate is a volatile liquid (Hawley, 1981) with a vapor pressure of 173 mm Hg at 20°C (Union Carbide, 1973). This suggests that evaporation from dry surfaces will be rapid. Since methyl acetate can volatilize from water (see Section 2.1.5.), evaporation from moist soil surfaces may also occur.

2.4. SUMMARY

When released to the aquatic environment, microbial degradation and volatilization are likely to be important removal mechanisms for methyl acetate. Based on limited experimental data and by analogy to ethyl acetate, methyl acetate appears to be susceptible to microbial degradation in the environment; U.S. EPA (1986a) suggested that methyl acetate should biodegrade moderately fast. The volatilization half-life from a river 1 m deep flowing at a speed of 1 m/sec with a wind velocity of 3 m/sec has been estimated to be 10.8 hours. Hydrolysis will not be important in neutral or acidic water, but may become significant in alkaline water; the respective hydrolysis half-lives at 25°C and pHs 7, 8 and 9 are 1.2 years, 45 and 4.5 days. Aquatic oxidation (by HO radical), adsorption to sediments and

bioconcentration are not expected to be important. If released to the atmosphere, methyl acetate will react with photochemically produced HO radical with a half-life of ~59 days at 19°C. If released to soil, methyl acetate is likely to be susceptible to significant leaching and biodegradation. Volatilization from dry surfaces is expected to be rapid.

3. EXPOSURE

Methyl acetate is a naturally occurring compound, which has been identified in volatile emissions from animal waste and forest fires (Graedel, 1978).

3.1. WATER

Methyl acetate was qualitatively detected in drinking water from Seattle, WA, as part of the National Organics Reconnaisance Survey (U.S. EPA, 1975). It has also been identified in wastewater effluents from chemical manufacturing and latex facilities in Louisville, KY (U.S. EPA, 1976). A total of 79 observation stations in the U.S. EPA STORET data base (gross analysis) have reported the detection of methyl acetate in concentrations ranging from 0-4500 mg/ℓ (U.S. EPA, 1986b).

3.2. FOOD

Pertinent data regarding the identification of methyl acetate in ready-to-eat foods could not be located in the available literature as cited in the Appendix.

3.3. INHALATION

Methyl acetate is predominantly used as a solvent in paint removers and lacquers (Wagner, 1978). Direct evaporation of this solvent into the atmosphere is probably the largest volume source of emission of methyl acetate to the environment. In addition to atmospheric emissions from solvents and lacquers, methyl acetate can also be emitted to the atmosphere during petroleum manufacturing (Graedel, 1978).

Methyl acetate was qualitatively detected in the air of the Talladega National Forest, AL (Holzer et al., 1977). It has also been detected in cigarette smoke in concentrations ranging from 0.02-14 µg/cigarette

depending upon the cigarette brand (Higgins et al., 1983). Approximately 1,003,761 U.S. workers are potentially exposed to methyl acetate according to a National Organic Hazard Survey (NIOSH, 1984).

3.4. DERMAL

Pertinent dermal monitoring data could not be located in the available literature as cited in the Appendix. Dermal exposure to methyl acetate used in paint removers and lacquers seems likely.

3.5. SUMMARY

Methyl acetate occurs naturally in volatile emissions from animal wastes and forest fires (Graedel, 1978). It has been qualitatively detected in drinking water from Seattle, WA (U.S. EPA, 1975) and in wastewater effluents from chemical manufacturing and latex facilities in Louisville, KY (U.S. EPA, 1976). Direct evaporation of methyl acetate solvent from paint removers and lacquers is probably the largest volume source of emission of the compound to the environment. Methyl acetate has been detected in the air of the Talledaga National Forest, AL (Holzer et al., 1977) and in cigarette smoke (Higgins et al., 1983).

4. PHARMACOKINETICS

Pertinent data regarding the pharmacokinetics and metabolism of methyl acetate are limited. After oral administration to rabbits, methyl acetate was hydrolyzed to methanol and acetic acid (Tambo, 1973). The alcohol caused drunkenness whereas the acetic acid produced acidosis in the blood. The administration of methyl acetate also decreased the pCO_2 and pO_2 in the blood. Inhalation of a paint thinner consisting of acetate esters including methyl acetate caused drunkenness and hallucination in human subjects (Tambo, 1973). Tada et al. (1974) exposed humans to 200 ppm (607 mg/m^3) methyl acetate, by inhalation, twice daily for 2 hours each for 3-4 days. Urinary excretion of methanol rose during the first daily exposure, peaked during the second exposure and returned to preexposure levels the following morning. Maximum urinary concentrations of methanol were generally >10 mg/ℓ. The authors have suggested that urinary concentration of methanol may serve as an indicator for methanol and methyl acetate exposure. It has been suggested that the methanol formed by hydrolysis in the body is responsible for the toxicity of metyl acetate (ACGIH, 1986).

5. EFFECTS

5.1. CARCINOGENICITY

Pertinent data regarding the carcinogenicity of methyl acetate could not be located in the available literature as cited in the Appendix, and this chemical is not scheduled for testing by the National Toxicology Program (NTP, 1986).

5.2. MUTAGENICITY

The only available studies found in the literature that address the mutagenicity of methyl acetate were conducted in the yeast Saccharomyces. Zimmermann et al. (1985) investigated the ability of methyl acetate to induce gene mutation or recombination as well as chromosome malsegregation (i.e., aneuploidy) in Saccharomyces cerevisiae at concentrations up to 3.85%. Methyl acetate was found to induce mitotic aneuploidy but not gene mutation or recombination. It might be hypothesized that methyl acetate interacts with target molecules that serve to ensure the proper segregation of chromosomes during cell division rather than interacting directly with DNA. Spindle proteins such as tubulin or microtubules would fall into this category. Studies by Groschel-Stewart et al. (1985) showed that methyl acetate does inhibit the assembly of porcine brain tubulin in vitro at a concentration slightly lower than what is needed to induce aneuploidy. Although methyl acetate does not appear to induce gene mutation and recombination in yeast, studies by Migliore et al. (1983) showed that it increased the induction of gene conversion in S. cerevisiae by cyclophosphamide.

5.3. TERATOGENICITY

Pertinent data regarding the teratogenicity of methyl acetate could not be located in the available literature as cited in the Appendix.

5.4. OTHER REPRODUCTIVE EFFECTS

Pertinent data regarding other reproductive effects of methyl acetate could not be located in the available literature as cited in the Appendix.

5.5. CHRONIC AND SUBCHRONIC TOXICITY

Pertinent data regarding the chronic and subchronic toxicity of methyl acetate could not be located in the available literature as cited in the Appendix.

5.6. OTHER RELEVANT INFORMATION

Munch (1972) listed an oral LD_{50} for methyl acetate of 50 mmol/kg (3705 mg/kg) in rabbits. As cited by von Oettinger (1960), Reus (1933) found that exposure to methyl acetate vapor at 19-21 mg/ℓ (6270-6930 ppm) for 6 hours/day for 7 days resulted in ocular irritation, salivation and progressive CNS depression in 4/5 cats. After 5 exposure days, one cat died from infiltration of the left pulmonary lobe and hyperemia. Three cats showed increased hemoglobin and erythrocyte values, two of which had a transient increase in leukocytes. Inhalation exposure of mice to 7900 ppm (24,000 mg/m³) for 459-480 minutes or cats to 9900 ppm (30,000 mg/m³) for 590 minutes, led to ocular irritation, salivation and dyspnea; however, all treated animals recovered after cessation of exposure (Reus, 1933). Concentrations of >20,000 ppm (>60,500 mg/m³) for ≤1 hour were lethal. Respiratory depression, vomiting and convulsions, characteristic of the early phases of a short-lived narcosis, were observed in mice exposed to 34 mg/ℓ (11,220 ppm) methyl acetate for 216 or 312 minutes and in cats exposed to 56 mg/ℓ (18,480 ppm) for 238 and 295 minutes. Application of 2 mℓ methyl acetate to rabbit skin produced erythema, whereas application of one drop to the rabbit eye led to hyperemia, edema and lacrimation (von Oettinger, 1960).

Fassett (1963) stated that the physiological effects of saturated aliphatic acetates included irritation to the mucous membranes and a slow onset of narcosis, with gradual recovery after cessation of exposure. Probably because of *in vivo* hydrolysis, exposure was accompanied by a great deal of acidosis.

As cited in Opdyke (1979), NIOSH (1977) listed the low toxic concentration of methyl acetate in man as 15,000 mg/m³. Humans exposed to 32,000 mg/m³ (10,000 ppm) continued to experience respiratory and mucous membrane irritation after cessation of exposure (Fassett, 1963). Reus (1933) reported that application of two drops to human skin caused cooling and desquamation but no irritation.

Methyl acetate, at a concentration of 5000 ppm (15,000 mg/m³), exerted an antihemolytic effect in a hypotonic rat erythrocyte medium (Holmberg et al., 1974). Abbondandolo et al. (1980) determined that methyl acetate was not cytolethal for *Schizosaccharomyces pombe* cells, but concentrations of 0.5-10.0% were highly lethal to V79 Chinese hamster cells in culture. A 5% concentration suppressed the aminopyrine demethylase activity in liver microsomes from both noninduced and phenobarbital-induced mice.

5.7. SUMMARY

There were no data available on the carcinogenic, reproductive, chronic or subchronic effects of methyl acetate administration. Acutely, the compound is a respiratory and mucous membrane irritant (Reus, 1933). In cats, inhalation exposure for 5 days to methyl acetate resulted in increased hemoglobin and erythrocyte levels, and a transient leukocytosis (Reus, 1933).

Toxic effects after acute inhalation exposure also included a gradual narcosis and metabolic acidosis (Fassett, 1963). Methyl acetate produced erythema when applied to rabbit skin, and hyperemia and edema when applied to the eye (von Oettinger, 1960). NIOSH (1977) stated that the lowest toxic concentration in man was 15,000 mg/m^3.

Zimmermann et al. (1985) found increases in mitotic aneuploidy and monosome formation in Saccharomyces cerevisiae colonies exposed to methyl acetate. Groschel-Stewart et al. (1985) showed that methyl acetate inhibits the assembly of porcine brain tublin in vitro. Abbondandolo et al. (1980) found that methyl acetate was more lethal to V79 Chinese hamster cells than to Schizosaccharomyces pombe cells in culture. In a hypotonic solution of rat erythrocytes, the ester exerted an antihemolytic effect (Holmberg et al., 1974).

6. AQUATIC TOXICITY

6.1. ACUTE

Munch (1972) reported threshold narcotic concentrations of 70-157 mmol/ℓ (5186-11,631 mg/ℓ) for tadpoles, Rana pipiens. Calmbacher (1978) reported that the 96-hour LC_{50} for bluegills, Lepomis macrochirus, was >100 mg/ℓ, although concentrations of ≥56 mg/ℓ caused abnormal behavior.

6.2. CHRONIC

Pertinent data regarding chronic toxicity of methyl acetate to aquatic organisms could not be located in the available literature as cited in the Appendix.

6.3. PLANTS

Pertinent data regarding effects of methyl acetate on aquatic plants could not be located in the available literature as cited in the Appendix.

6.4. RESIDUES

Pertinent data regarding methyl acetate residues in aquatic biota could not be located in the available literature as cited in the Appendix.

6.5. SUMMARY

The only information available concerning the effects of methyl acetate on aquatic organisms was provided by Munch (1972), who reported threshold narcotic concentrations of 70-157 mmol/ℓ (5186-11,631 mg/ℓ) for tadpoles, Rana pipiens, and by Calmbacher (1978), who reported that the 96-hour LC_{50} for bluegills, Lepomis macrochirus, was >100 mg/ℓ.

7. EXISTING GUIDELINES AND STANDARDS

7.1. HUMAN

The ACGIH (1985-1986) adopted a TLV-TWA of 200 ppm (~610 mg/m^3) and a STEL of 250 ppm (~760 mg/m^3) for methyl acetate. The TLV, which is the same as that for methanol, was based upon the *in vivo* hydrolysis of methyl acetate to methanol, and the subsequent toxicity of the latter compound (ACGIH, 1986). The committee claimed that no cases of irritation or systemic injury had ever been reported in workers exposed to concentrations <200 ppm. The OSHA (1985) PEL is 200 ppm.

7.2. AQUATIC

Guidelines and standards for the protection of aquatic biota from the effects of methyl acetate could not be located in the available literature as cited in the Appendix.

8. RISK ASSESSMENT

There were no available data on the carcinogenic, reproductive, chronic or subchronic effects of methyl acetate administration. Acutely, the compound is a respiratory and mucous membrane irritant (Reus, 1933). In cats exposed for 5 days to the compound by inhalation, Reus (1933) found increased hemoglobin and erythrocyte levels, and a transient leukocytosis. Toxic effects after acute exposure also included a gradual narcosis and metabolic acidosis (Fassett, 1963). Methyl acetate produced erythema when applied to rabbit skin, and hyperemia and edema when applied to the eye (von Oettinger, 1960). NIOSH (1977) stated that the lowest toxic concentration in man was 15,000 mg/m^3.

Zimmermann et al. (1985) found increases in chromosomal aneuploidy and monosome formation in S. cerevisiae colonies exposed to methyl acetate. Abbondandolo et al. (1980) found that methyl acetate was more lethal to V79 Chinese hamster cells than to S. pombe cells in culture. In a hypotonic solution of rat erythrocytes, the ester exerted an antihemolytic effect (Holmberg et al., 1974).

Since methyl acetate hydrolyzes to methanol and acetate in vivo and since the toxic effects are attributed to methanol (ACGIH, 1986), it is appropriate to derive an RfD for methyl acetate by analogy to methanol. An RfD of 0.5 mg/kg/day for methanol was recently verified by the U.S. EPA (1986c). The RfD was based on a NOEL of 500 mg/kg/day of methanol administered by gavage for 90 days to Sprague-Dawley rats (Toxicity Research Laboratory, 1986) and the application of an uncertainty factor of 1000. At 500 mg/kg/day, there were no effects on body weight gain, food consumption, moribundity, mortality, clinical chemistry, ophthalmological evaluations or

gross and microscopic evaluations. The next higher dose tested, 2500 mg/kg/day, which was considered to be the LOAEL, was associated with elevated levels of SGPT, serum alkaline phosphatase and increased liver weights. Multiplying the NOEL for methanol of 500 mg/kg/day by the ratio of the molecular weights of methyl acetate to methanol (74.08/32.04) results in a NOEL for methyl acetate of 1156 mg/kg/day. Dividing by an uncertainty factor of 1000 (10 for interspecies extrapolation, 10 to protect sensitive individuals and 10 to approximate chronic exposures) results in an RfD for methyl acetate of 1 mg/kg/day or 81 mg/day for a 70 kg man. This RfD value is recommended until further, more definitive data concerning the carcinogenicity, reproductive, subchronic or chronic toxicity of methyl acetate become available.

9. REPORTABLE QUANTITIES

9.1. REPORTABLE QUANTITY (RQ) RANKING BASED ON CHRONIC TOXICITY

Data are not sufficient for deriving an RQ (Table 9-1).

9.2. WEIGHT OF EVIDENCE AND POTENCY FACTOR ($F=1/ED_{10}$) FOR CARCINOGENICITY

Data were insufficient for derivation of a carcinogenic potency factor. Since there are no data regarding the carcinogenicity of methyl acetate to laboratory animals or humans, this compound is most appropriately classified as an EPA Group D chemical (U.S. EPA, 1986d).

TABLE 9-1

Methyl Acetate

Minimum Effective Dose (MED) and Reportable Quantity (RQ)

Route:	
Dose:	
Effect:	
Reference:	
RV_d:	
RV_e:	
Composite Score:	
RQ:	Data are not sufficient for deriving an RQ.

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APPENDIX

LITERATURE SEARCHED

This profile is based on data identified by computerized literature searches of the following:

GLOBAL
TSCATS
CASR online (U.S. EPA Chemical Activities Status Report)
CAS online STN International
TOXLINE
TOXBACK 76
TOXBACK 65
RTECS
OHM TADS
STORET
SRC Environmental Fate Data Bases
SANSS
AQUIRE
TSCAPP
NTIS
Federal Register

These searches were conducted in April, 1986. In addition, hand searches were made of Chemical Abstracts (Collective Indices 6 and 7), and the following secondary sources were reviewed:

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